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# Interpretation of Blood Gas and Acid-Base Data

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# Interpretation of Blood Gas and Acid-Base Data

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Before discussing clinical cases we would like to present a brief recent history of developments in the area of acid-base and blood gas analysis so some appreciation of the efforts of those who have worked to simplify measurement techniques and clarify clinical evaluation procedures may be justly given. We would also like to illustrate with the aid of two clinical cases the importance of proper interpretation of acid-base and blood gas data.

In 1908 Henderson applied physico-chemical principles to estimation of the hydrogen ion concentration in physiologic fluids. He stated that

$$[H] = K \cdot \frac{[acid]^1}{[base]}$$

In this equation he laid down the foundation for the relationship between an excess of acid or base in the blood and an increase or decrease in the hydrogen ion concentration.

Hasselbalch modified the original equation by converting it to logarithmic terms.

$$pH = pK + \log \frac{[acid]^1}{[base]}$$

Hasselbalch was the first to measure blood pH in 1910.[1]

The principles and measurement of acid-base and blood gas phenomena have been nurtured and refined over the past fifty years.

The first measurements of acid-base and blood gas parameters were performed on clinical patients in the early 1950's. This significant step contributed to the survival of numerous patients whose treatment (life) had previously depended on mere guesswork on the part of the clinician.

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Fortuitously for veterinarians and dogs, in particular, most of the research in the area of acid-base balance and blood gas analysis has revolved around the canine species. Prediction equations for this species are applied to humans.

When dealing with pathological disturbances we often tend to forget two very basic facts of life for terrestrial species. The first is that multicellular species are faced with the minute-by-minute defense of their acid-base balance against acid loads of metabolism. The second is that normal osmolality must be maintained in a dessicating environment. We see homeostasis of acid-base balance and osmolality as two of an animal's three most urgent needs: the third being availability of oxygen.

The cause of death in all animals is due to failure of the regulatory mechanisms in one or more of the above systems. For example, in diabetes mellitus, death ensues because of the acidosis and hyperosmolality that are the result of insulin deficiency. Furthermore, in the treatment of this disease, the activity of insulin is serverely compromised by the acidosis.

Acid-base and blood gas values are normally confined within narrow, stable limits. Mechanisms controlling these values are finely tuned. As compared to blood glucose, which in a dog may vary between 50 mg/dl and 200 mg/dl without much of a deleterious effect on the animal, even a twofold change in $[HCO_3^-]$ is extremely serious.

### *Case 1*

The following values were taken half an hour before death and prior to the administration of bicarbonate to a dog undergoing surgery for correction of patent ductus arteriosus.

| Values | Normal Range* |
|---|---|
| pH = 7.128 | pH = 7.437-7.469 |
| $pO_2$ = 59.9 | $pO_2$ = 87-95[2] |
| $pCO_2$ = 81.5 | $pCO_2$ = 34-38[2] |
| $HCO_3^-$ = 26.5 | $HCO_3^-$ = 24-26[2] |
| $TCO_2$ = 29.0 | |
| BE = −1.1 | BE = −4.5-+5.0[4] |
| Hb = 3.6 | Hb = 12-18[5] |
| Osmolality = 316 | Osmolality = 285-292[3] |
| $Cl^-$ = 70 | $Cl^-$ = 105-113[2] |

***Evaluation***

The first step in evaluation of data is to decide if the data is real or if some error has lead to misfitting data. Check can be made of blood gas data using the following formula:

$$[H+] = \frac{24 \times pCO_2}{[HCO_3^-]}$$[6]

$$[H^+] = \frac{24 \times 815}{26.5}$$

$$74.47^* \approx 73.8 \text{ nmoles/l}$$

We find 74.47 approximately equals 73.8 nmoles/l and so no gross errors have been made in sampling or measurement.

The next steps in evaluation should be an orderly inspection of the values.

**pH**—A pH of 7.128 is a severe acidosis but does not reveal the cause of the acidosis. A pH value is of no use to the clinician in formulating treatment because it does not show whether the condition is metabolic or respiratory, whether there is any compensation, nor does it help in identification of whether the condition is acute or chronic.[7]

**$pO_2$**—A low value is reflected for $O_2$ dissolved in the plasma so we may conclude that the animal is hypoxic. The degree of hypoxia is not a mere reflection of this absolute value. Rather, it depends on the body temperature, pH, 2,3 DPG concentration, and the position of the oxygen dissociation curve. Low body temperatures reduce dissociation between $0_2$ and Hb; high body temperatures act conversely. A low pH (acidosis) causes problems because the degree of saturation of Hb is decreased; high pH causes Hb to be saturated at a much lower partial pressure of oxygen—a problem can be caused at the tissues because the gradient for $O_2$ is not steep enough to allow $O_2$ to leave the blood for the tissues. Consequently, the tissues have to be abnormally low in $O_2$ before there is any delivery of $O_2$ from the blood. Due to the low pH in this instance and the low $pO_2$, the degree of saturation was 81.2%. As this condition progresses hypoxia will lead to anaerobic metabolism and a late metabolic acidosis will be superimposed on the primary respiratory acidosis.

The conditions mentioned above are responsible for shifting the oxygen dissociation curve to the left or right of its normal position. They are detected when a $P_{50}$ value is calculated.

**$pCO_2$**—Severe hypercarbia is present. The severity of the hypercarbia makes it a primary condition. Circumstances are such that voluntary lowering of the ventilation to the extent seen here would produce fatal hypoxia and $CO_2$ narcosis.[8] A prediction equation allows us to verify this premise.[9]

$$\Delta [H^+] = 0.8\, \Delta\, pCO_2$$
$$34.47 = 0.8 \times 41.5$$
$$34.47 \approx 33.2$$

**$[HCO_3^-]$**—The bicarbonate concentration lies slightly above the normal range.[2] The reason for the increase is due to a shift of the carbonic acid equilibrium to the right. Such a shift normally occurs to the extent of 3-4 mEq/1 with respiratory acidosis due to $CO_2\uparrow + H_20 \rightleftharpoons H + HCO_3^-\uparrow$.

**$TCO_2$**—The total $CO_2$ will mostly lead the $[HCO_3^-]$ by a small amount, as under nearly all circumstances the $CO_2$ is derived from the $[HCO_3^-]$. (Total $CO_2$ includes all forms of $CO_2$ in the blood.)

**BE**—This value shows the amount of titratable acidity present in the blood. Because it was an experimentally derived value, it is directly applied to correction of a metabolic disturbance therapeutically. In this case the disturbance is entirely respiratory and would not respond to correction by $NaHCO_3$ therapy.

**Hb**—Severe anemia is present.

**Osmolality**—The dog is hyperosmolar[3] but the reason for its hyperosmolality was not determined because of lack of data. The serum $Na^+$, glucose, and BUN all must be known before pathogenesis can be determined. Treatment for the acid-base disorder would be worked out on the basis of dealing with a concurrent hyperosmolality disorder.

*95% confidence.

**$Cl^-$**—A hypochloremia[2] is present. Since the bicarbonate is normal, an unmeasured anion could account for the measured value. (Body fluids are kept close to neutral pH so when a loss of an anion such as chloride occurs there must be a corresponding increase in another anion.) On the other hand an unmeasured osmole could also account for the low chloride.

**Pathologists' Diagnosis**—Acute uncompensated respiratory acidosis with concurrent hypochloremia, hyperosmolality, and anemia.

**Therapy**—The dog was given bicarbonate.

**Result of Treatment**—Ventricular fibrillation occurred after therapy and the dog died.

**Reason for Treatment Failure**—The dog's problem was not one of metabolic acidosis but was one of severe respiratory acidosis. The rapid infusion of bicarbonate led to a fatal cardiac arrhythmia.[6]

**Correct Treatment**—The treatment this animal should have received was hyperventilation to blow off the $CO_2$ and raise the oxygen, followed by a transfusion and supplemental laboratory data to determine the causes of hypochloridemia and hyperosmolality.

### *Case 2*

Blood gas samples were taken from a dog that had undergone concurrent surgery for a diaphragmatic hernia and caesarian section. Samples were taken at the termination of anesthesia. The surgeon suspected the dog was not recovering due to blood loss and ordered 500 ml of blood to be given.

| Values |
|---|
| pH = 6.867 |
| $pO_2$ = 528.3 |
| $pCO_2$ = 29.9 |
| $HCO_3^-$ = 5.2 |
| $TCO_2$ = 6.3 |
| BE = −29.4 |
| Hb = 3.0 |

### *Evaluation*

A check of the data shows there has been no serious sampling or measurement errors.

$$[H^+] = \frac{24 \times pCO_2}{HCO_3^-}$$

$$135.83 \approx 138$$

**pH**—Severe acidosis is present. The limits of life are defined by pH 7.00 and pH 7.7.[10]

**$pO_2$**—Positive pressure ventilation during surgery has caused this hyperoxia.

**$pCO_2$**—Ventilation with 100% oxygen is responsible for the washout effect on $pCO_2$ and resultant hypocarbia. A decreased $pCO_2$ is seen when compensation for a metabolic acidosis is present but this was not the case here: the animal's ventilation was controlled by the anesthesiologist. We must conclude that the respiratory alkalosis was iatrogenic.

**$[HCO_3^-]$**—A very severe deficit exists indicating a severe metabolic acidosis is present.

**BE**—A deficit in fixed base or an excess of hydrogen ions to the tune of 29.4 mmole/l of bicarbonate space is present.

**Pathologist's Diagnosis**—Hyperoxia, severe metabolic acidosis, respiratory alkalosis, anemia.

**Treatment**—The dog was given 500 ml of blood post-surgically.

**Result of Treatment**—The dog was removed from the assisted ventilation and died 15 minutes postsurgically.

**Reason for Treatment Failure**—This dog had no reason to want to breathe; its $pCO_2$ was below normal range; its $pO_2$ was well above normal arterial oxygen. Infusion of blood would add to the metabolic acidosis already present and may have overloaded the circulatory system which was already depressed due to the effect of acidosis on the myocardium.[8,11]

### *Conclusion*

The correct functioning of cellular metabolism depends on the acid-base balance of the animal. Partition and distribution of drugs used is dependent on the acid-base balance of the animal. The cases illustrated above show the result of misinterpretation of acid-base data. Neither of the cases is presented as typical for that particular set of circumstances—it is not possible to guess at the acid-base values an animal may have present.

*$[H^+] = 10^{-pH}$

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The VCII Women's 3-player intramural volleyball team placed first in class A competition during winter quarter.

The VMII men's basketball team finished the recent intramural season with a record of 7-1.

ST. PAUL, Minn. — A treatment technique commonly used for cats with urethral obstruction may cause more harm than good claim two veterinary scientists at the University of Minnesota. The incidence and features of bacterial urinary infections caused by indwelling urethral catheterization of normal cats are being investigated by Carl Osborne, D.V.M., Ph.D., and George Lees, D.V.M., in a study funded by Morris Animal Foundation.

Preliminary results of the study indicate that an inflammatory reaction is caused in the urethral tissues by catheters and that bacterial infection of urine in the bladder often develops during catheterization. The urethral response is a "foreign body reaction" which may be damaging to the urethra especially when infection develops as well.

Urinary infections which developed during the study were of variable severity. Unexpectedly, the more severe infections occurred in cats given fluid therapy similar to that which is often given to obstructed cats. Administration of fluids increases volume of urine production and decreases urine concentration, an effect called diuresis. It was expected that diuresis would be beneficial by having a "washing-out" effect on the urinary bladder. Although diuresis increased urine flow through the urinary tract, rather than being beneficial this effect appeared to be associated with increased susceptibility to infection.

"The reason for this phenomenon," explained Dr. Lees, "appears to be that cat urine is normally so concentrated that bacterial growth in it is inhibited. By diluting the urine, diuresis increases the susceptibility to infection by making the urinary tract a more favorable place for bacteria to flourish," he continued.

Although antibiotic therapy tended to offset bacterial infections during catheterization, it did not prevent the problem entirely. Drs. Lees and Osborne feel that the study has demonstrated that indwelling urethral catheterization often induces bacterial infections in cats, that diuresis during catheterization increases the risk of severe infection, and that antibiotic therapy will not reliably prevent catheter-induced infections.

Two articles resulting from this study will be published in professional journals in 1979.